A 43-year-old obese woman presents to the emergency room with severe epigastric pain that started after a heavy meal. She has had several milder episodes before related to fatty food consumption. Physical examination reveals marked tenderness in the right subcostal area. Which of the following hormones most likely provoked the current attack in this patient?

A. Secretin

B. Cholecystokinin

C. Gastrin

D. Vasoactive intestinal peptide (VIP)

E. Motilin

F. Somatostatin

Submit

A 43-year-old obese woman presents to the emergency room with severe epigastric pain that started after a heavy meal. She has had several milder episodes before related to fatty food consumption. Physical examination reveals marked tenderness in the right subcostal area. Which of the following hormones most likely provoked the current attack in this patient?

- A. Secretin (2%)
- ✓ B. Cholecystokinin (89%)
- C. Gastrin (5%)
- D. Vasoactive intestinal peptide (VIP) (1%)
- E. Motilin (0%)
- F. Somatostatin (0%)

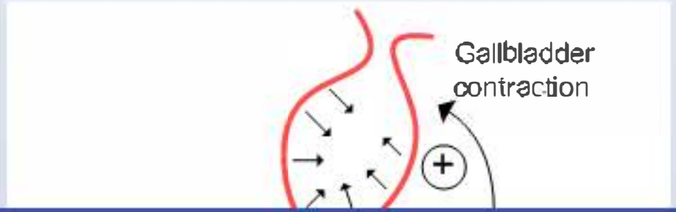
Omitted
Correct answer
B

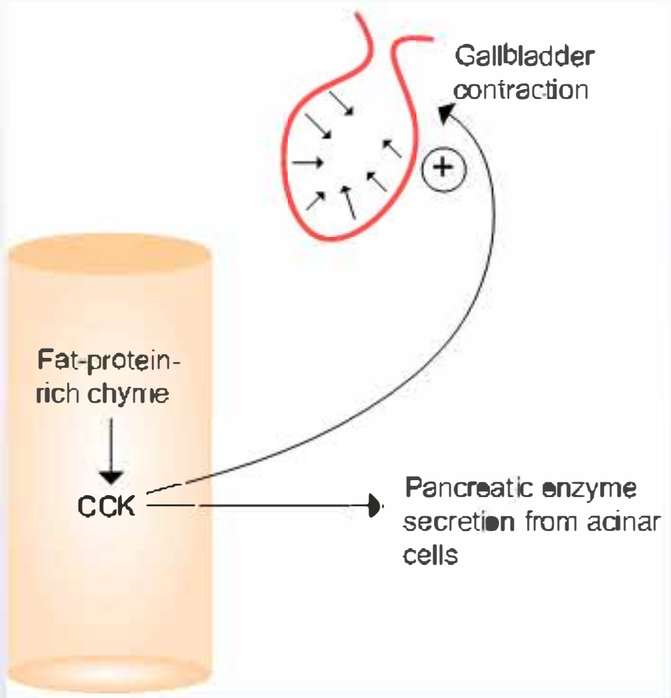
89%
Answered correctly

36 secs
Time Spent

02/28/2020
Last Updated

Explanation





This patient presents with classic signs and symptoms of biliary colic. Moreover, she has three well-known risk factors for gallbladder disease ("forty," "fat," and "female"). Cholecystokinin (CCK) is the hormone responsible for gallbladder contraction. It is produced by I cells of the duodenum and jejunum when fat-protein-rich chyme enters the duodenum. It functions to increase pancreatic enzyme secretion (by acinar cells) and gallbladder contraction, and to decrease gastric emptying. In cholecystitis, fatty foods increase CCK production and pain occurs when an inflamed and/or obstructed gallbladder contracts.

(Choice A) Secretin promotes bicarbonate secretion from pancreatic ductal epithelium. It has no effect on pancreatic enzyme secretion.

(Choice C) Gastrin is a hormone made in the stomach in response to stomach distention, peptides, and vagal stimulation. It stimulates gastric acid secretion and motility.

(Choice D) Vasoactive intestinal peptide (VIP) is produced in the pancreas and stimulates intestinal water secretion, counteracts gastrin in the stomach, and promotes bicarbonate secretion for the pancreas.

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(Choice E) Motilin is a hormone made in the small intestine that promotes intestinal motility.

(Choice F) Somatostatin is a hormone made in numerous tissues (hypothalamus, stomach, intestine, pancreas) in response to low pH. Its production is inhibited by vagal stimulation. Somatostatin inhibits the release of growth hormone (GH) and thyroid stimulating hormone (TSH), and suppresses the release of myriad other molecules (e.g. gastrin, CCK, VIP, secretin, insulin, glucagon).

Educational objective:
Cholecystokinin (CCK) is the hormone responsible for gallbladder contraction. It is made in the duodenum and jejunum in response to fatty acids and amino acids.

Physiology	Gastrointestinal & Nutrition	Cholecystitis
Subject	System	Topic

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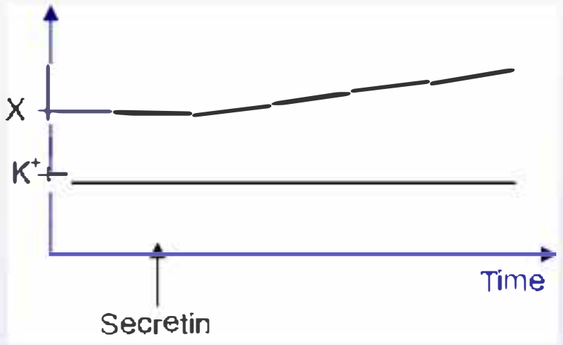
In an animal experiment, the pancreatic duct is cannulated and different secretory stimuli are applied to the gland. The following tracings are obtained by measuring pancreatic fluid constituents:



Substance 'X' is most likely which of the following?

A. Sodium
B. Chloride
C. Trypsinogen
D. Bicarbonate
E. Phosphate

Submit

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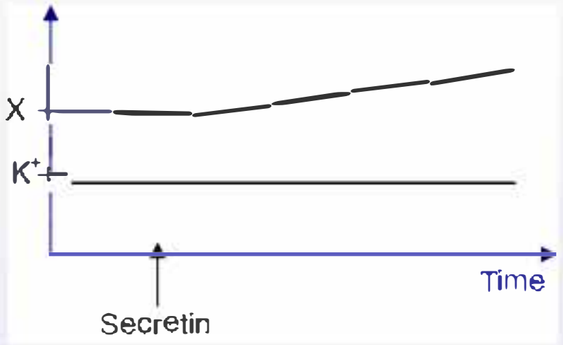

Substance 'X' is most likely which of the following?

A. Sodium (1%)

B. Chloride (3%)

C. Trypsinogen (13%)

✓ D. Bicarbonate (80%)

E. Phosphate (0%)

Omitted
Correct answer
D

80%
Answered correctly

02 secs
Time Spent

02/03/2020
Last Updated

Omitted
Correct answer
D

80%
Answered correctly

02 secs
Time Spent

02/03/2020
Last Updated

Explanation

Secretin is a hormone produced by duodenal S-cells and released in response to increased duodenal H^+ concentrations. Secretin stimulates pancreatic ductal cells to increase bicarbonate secretion in order to neutralize the acidity of the gastric contents entering the duodenum. Remember that pancreatic juice is an isotonic secretion, which normally contains Na^+ and K^+ in the same concentrations as found in plasma, a higher HCO_3^- concentration than in plasma and a lower Cl^- concentration than in plasma. As pancreatic juice flow rates and secretin stimulation increase, the concentration of HCO_3^- increases and the concentration of Cl^- decreases.

(Choice A) Regardless of flow rate or hormonal stimulation, the sodium concentration of pancreatic secretions is identical to that of plasma.

(Choice B) The chloride content of pancreatic secretions decreases as the bicarbonate content increases, because chloride and bicarbonate are exchanged for one another at the apical surfaces of pancreatic ductal cells.

(Choice C) Trypsinogen is a pancreatic proenzyme that is converted to its active form, trypsin, by enterokinase in the intestinal brush border. Active trypsin can also activate trypsinogen. Cholecystokinin and cholinergic stimulation increase the secretion of pancreatic enzymes.

(Choice E) Phosphate secretion is not regulated in the pancreas.

Educational Objective:
Duodenal S-cells secrete secretin in response to increasing H^+ concentrations. Secretin increases pancreatic bicarbonate secretion. The chloride content of pancreatic secretions decreases in proportion to bicarbonate concentration increases.

Physiology	Gastrointestinal & Nutrition	Pancreas physiology
Subject	System	Topic

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A 32-year-old woman comes to the office due to 6 months of persistent diarrhea. She has had 8-10 episodes a day of tea-colored, odorless, watery stools. The symptoms have persisted despite a lactosefree diet and a fasting trial. The patient has had no abdominal pain, fever, or vomiting. Temperature is 36.7 C (98 F). Abdominal examination is unremarkable. Sampling of her gastric contents shows a total lack of gastric acid secretion. Somatostatin treatment promptly relieves the symptoms. An excess of which of the following hormones is most likely responsible for this patient's symptoms?

A. Cholecystokinin
B. Gastrin
C. Ghrelin
D. Glucagon
E. Motilin
F. Vasoactive intestinal peptide

Submit

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- A. Cholecystokinin (15%)
- B. Gastrin (8%)
- C. Ghrelin (2%)
- D. Glucagon (3%)
- E. Motilin (7%)
- ✓ F. Vasoactive intestinal peptide (63%)

Omitted
Correct answer
F

63%
Answered correctly

02 secs
Time Spent

04/04/2020
Last Updated

Explanation

This patient has secretory diarrhea, distinguished from inflammatory diarrhea by the absence of blood or pus and from osmotic diarrhea (eg, due to lactose intolerance) by its failure to improve with dietary modification. Excess **vasoactive intestinal peptide** (VIP) secretion due to a pancreatic islet cell tumor called VIPoma can result in **watery diarrhea, hypokalemia, and achlorhydria** (WDHA) syndrome (**pancreatic cholera**). VIP stimulates pancreatic bicarbonate and chloride secretion, and its binding to intestinal epithelial cells leads to adenylate cyclase

This patient has secretory diarrhea, distinguished from inflammatory diarrhea by the absence of blood or pus and from osmotic diarrhea (eg, due to lactose intolerance) by its failure to improve with dietary modification. Excess **vasoactive intestinal peptide** (VIP) secretion due to a pancreatic islet cell tumor called VIPoma can result in **watery diarrhea, hypokalemia, and achlorhydria** (WDHA) syndrome (**pancreatic cholera**). VIP stimulates pancreatic bicarbonate and chloride secretion, and its binding to intestinal epithelial cells leads to adenylate cyclase activation and increased cyclic AMP production, causing sodium, chloride, and water secretion into the bowel (**secretory** watery **diarrhea**, often >3 L/day).

Somatostatin (octreotide) decreases the production of many gastrointestinal (GI) hormones (eg, VIP, gastrin, glucagon, cholecystokinin [CCK]). Its inhibition of VIP production by this VIPoma is responsible for the resolution of this patient's symptoms.

(Choice A) CCK causes increased secretion of pancreatic enzymes and bicarbonate, gallbladder contraction, and inhibition of gastric emptying. It does not cause WDHA syndrome. CCK is produced by I cells of the proximal small bowel mucosa in response to fatty acids and amino acids.

(Choice B) Gastrin is produced by G cells in the stomach mucosa. It stimulates gastric acid production and growth of the gastric mucosa. Gastrinomas classically cause intractable peptic ulcer disease (Zollinger-Ellison syndrome), not WDHA syndrome.

(Choice C) Ghrelin is produced in the stomach and regulates food intake. Levels increase in fasting states and fall after eating. Ghrelin antagonists are being investigated as drug targets.

(Choice D) A glucagonoma is a pancreatic alpha cell tumor that hypersecretes glucagon and can cause secondary diabetes mellitus and necrolytic migratory erythema of the skin.

(Choice E) Motilin is produced by the duodenal mucosa and stimulates smooth muscle contraction in the upper GI tract. The antibiotic erythromycin acts as an agonist at motilin receptors in the stomach and duodenum, contributing to the drug's GI side effects.

Educational objective:
VIPomas are pancreatic islet cell tumors that hypersecrete vasoactive intestinal peptide (VIP), which increases intestinal chloride loss into the stool and causes excess losses of the accompanying water, sodium, and potassium. VIP also inhibits gastric acid secretion. Somatostatin inhibits the secretion of VIP and is used to treat the symptoms of VIPoma.

References

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References

- Sporadic pancreatic vasoactive intestinal peptide-producing tumor (VIPoma) in a 47-year-old male.
- The physiological roles of secretin and its receptor.

Physiology	Gastrointestinal & Nutrition	Vipoma
Subject	System	Topic

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A 62-year-old man comes to the office for evaluation of abdominal swelling. Over the last few weeks his abdomen has become more distended with associated mild discomfort and decreased appetite. Past medical history includes multiple admissions for alcohol-induced pancreatitis. The patient currently drinks 1 pint of liquor daily and does not use tobacco or illicit drugs. Physical examination reveals mildly icteric sclera and multiple spider angiomas on the upper chest. The abdomen is distended and there is dullness to percussion that changes location when the patient lays on either side. Serum sodium is 130 mEq/dL and creatinine is 1.1 mg/dL. Which of the following would most likely be seen in this patient?

	Serum antidiuretic hormone	Urine sodium	Total body volume
A.	↑	↓	↑
B.	↑	↓	↓
C.	↑	↑	normal
D.	↓	↑	normal
E.	↑	↑	↓

Submit

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	Serum antidiuretic hormone	Urine sodium	Total body volume
✓ A. (62%)	↑	↓	↑
B. (3%)	↑	↓	↓
C. (20%)	↑	↑	normal
D. (8%)	↓	↑	normal
E. (4%)	↑	↑	↓

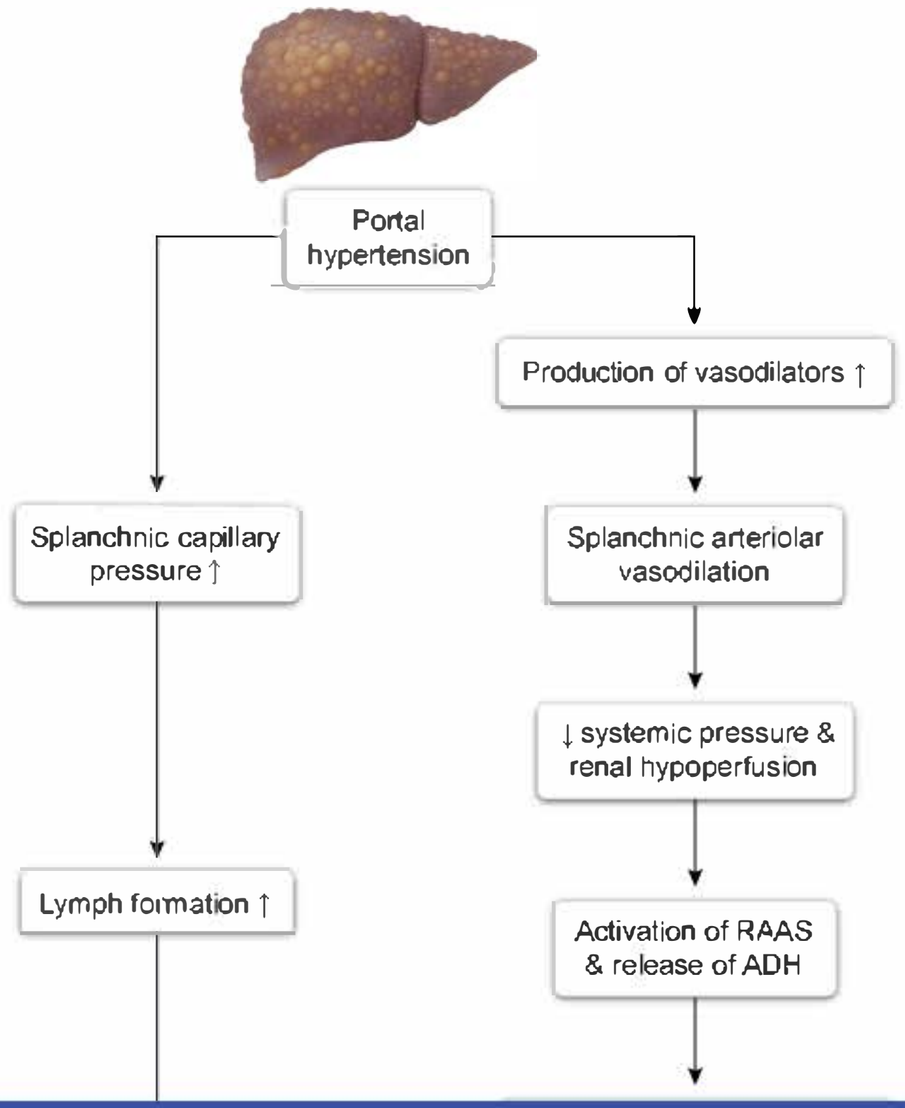
Omitted
Correct answer
A

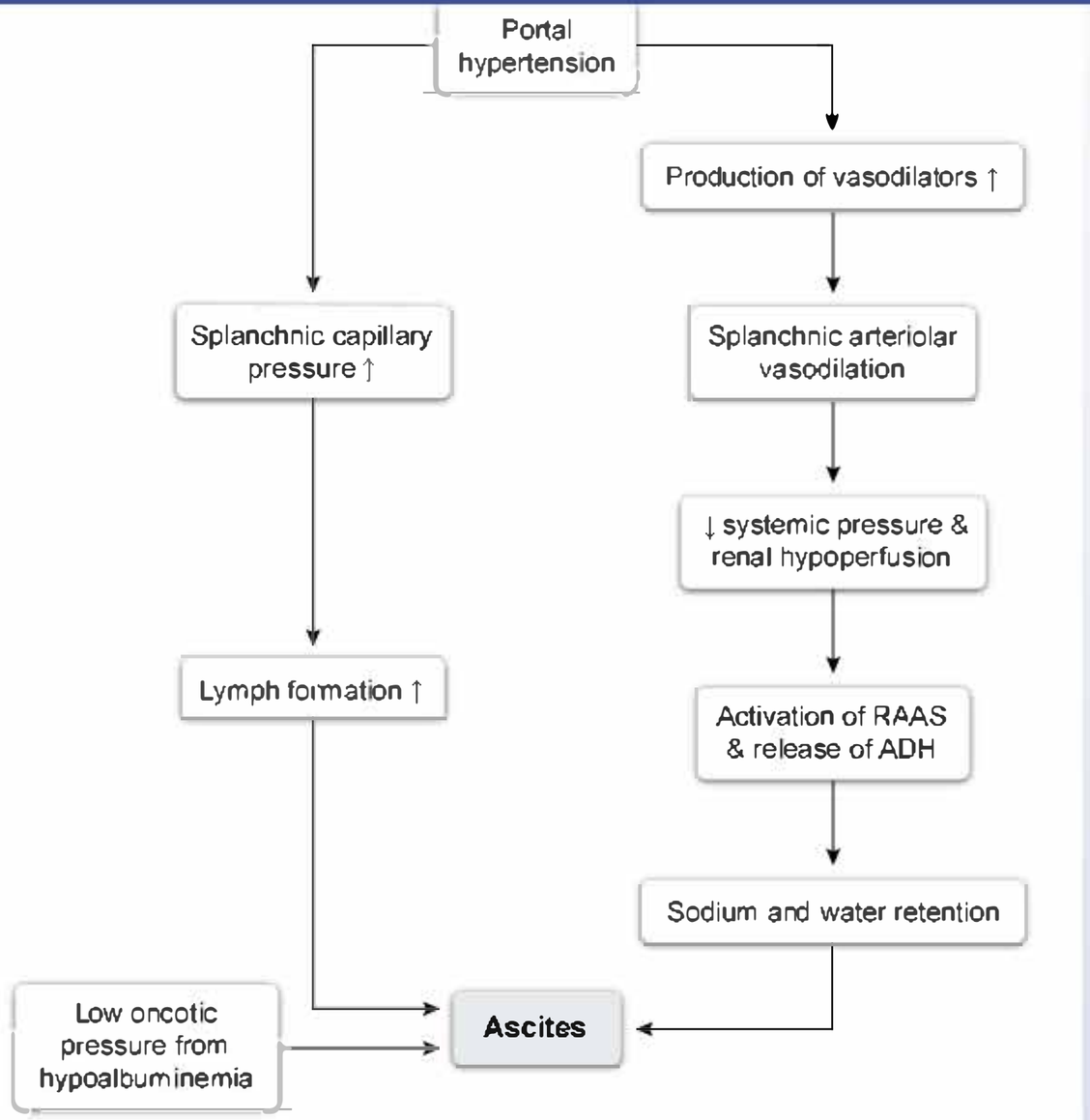
62%
Answered correctly

04 secs
Time Spent

03/31/2020
Last Updated

Pathogenesis of ascites in cirrhosis




Portal hypertension
Production of vasodilators ↑
Splanchnic capillary pressure ↑
Splanchnic arteriolar vasodilation
↓ systemic pressure & renal hypoperfusion
Lymph formation ↑
Activation of RAAS & release of ADH
Sodium and water retention
Low oncotic pressure from hypoalbuminemia
Ascites

ADH = antidiuretic hormone; RAAS = renin-angiotensin-aldosterone system.

This patient with a history of heavy alcohol use, abdominal swelling with shifting dullness to percussion (indicative of intraabdominal fluid), and stigmata of chronic liver disease (eg, spider angiomas, jaundice) has **ascites** due to cirrhosis. **Cirrhosis** occurs from progressive hepatic fibrosis, resulting **portal hypertension** due to increased sinusoidal blood flow resistance. There is also increased formation of vasodilatory factors (eg, nitric oxide), possibly due to bacterial products (eg, endotoxins) that accumulate because of reduced host defenses and increased portosystemic shunting. This results in the following hemodynamic alterations:

- Reduced systemic vascular resistance along with blood pooling in the splanchnic vascular bed leads to reduced blood pressure. Heart rate and cardiac output increase to compensate (hyperdynamic circulation).
- Reduced renal perfusion leads to activation of the reninangiotensin-aldosterone system (RAAS) and **increased release of antidiuretic hormone (ADH)** in an effort to restore circulating volume.
- The combined aldosterone and ADH effects lead to **increased renal resorption of sodium** (ie, lower urine sodium) and water.

Although this restores renal perfusion, the resistance to splanchnic flow and hyperdynamic circulation promotes third-spacing of fluids into the abdominal cavity (ie, ascites) and interstitium (ie, edema). This is compounded by low serum oncotic pressure due to hypoalbuminemia from liver synthetic dysfunction. Therefore, despite **increased total body volume**, patients with cirrhosis remain intravascularly depleted and continue to release hypovolemic hormones. Eventually, the resultant elevated ADH secretion can lead to **hypervolemic hyponatremia**, which is common in patients with advanced cirrhosis.

(Choices B, C, D, and E) Splanchnic vasodilation results in renal hypoperfusion, stimulating increased ADH secretion and activation of the RAAS. This leads to increased renal resorption of sodium, lowering the urinary sodium concentration. Third-spacing of fluid results in increased total body volume.

Educational objective:
Portal hypertension in cirrhosis leads to vasodilation and decreased systemic perfusion pressure, which causes antidiuretic hormone release and activation of the renin-angiotensin-aldosterone system, promoting sodium and water retention. However, due to resistance to splanchnic flow, low oncotic pressure, and hyperdynamic circulation, the fluid is third-spaced into the extravascular compartments (eg, ascites). Therefore, despite increased total body volume, patients with cirrhosis remain intravascularly volume depleted.

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References

- Hyponatremia in cirrhosis: pathophysiology and management.

Physiology	Gastrointestinal & Nutrition	Cirrhosis
Subject	System	Topic

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pressure from hypoalbuminemia → **Ascites**

ADH = antidiuretic hormone; **RAAS** = renin-angiotensin-aldosterone

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Educational objective:
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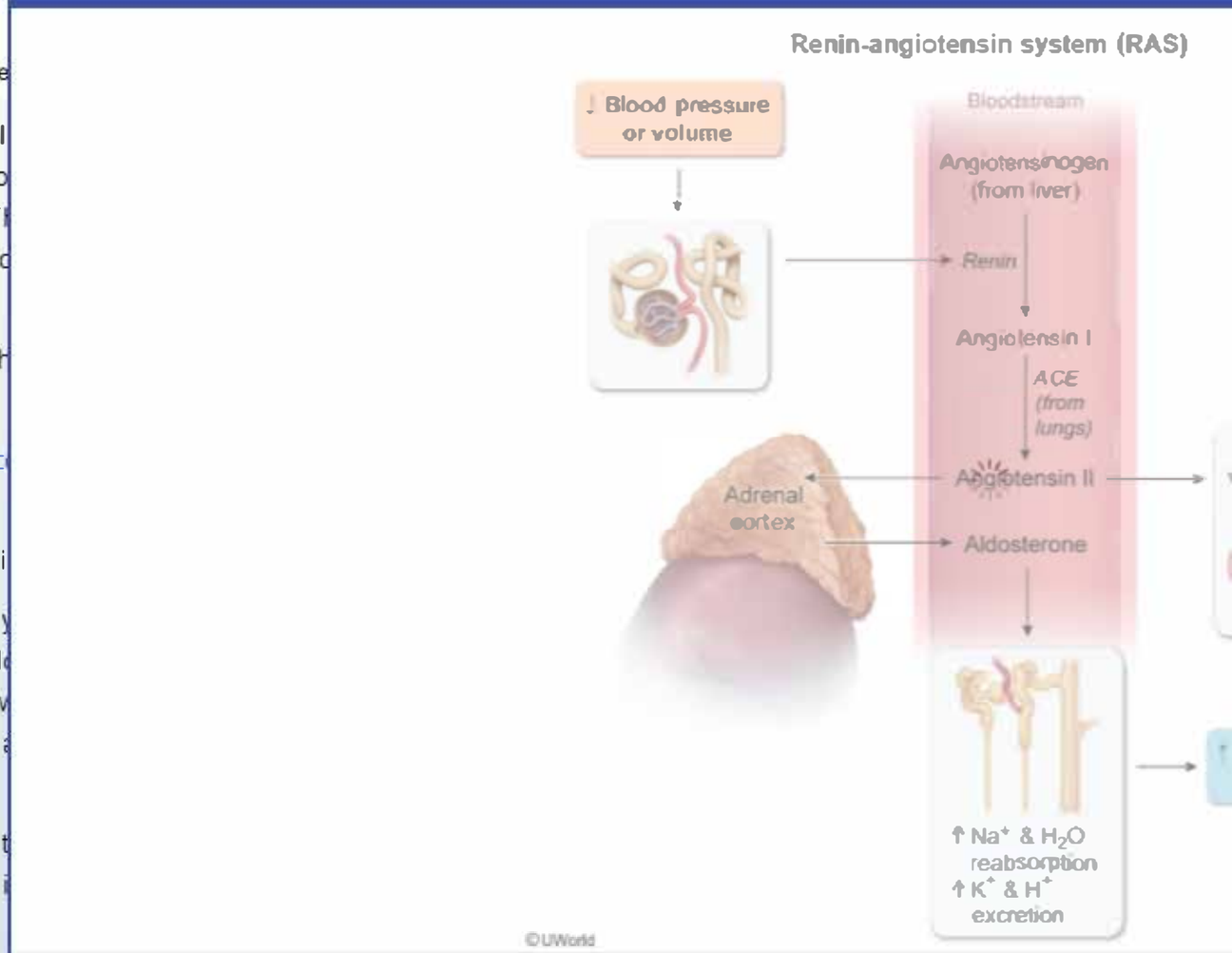

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pressure from hypoalbuminemia → Ascites

ADH = antidiuretic hormone; RAAS = renin-angiotensin-aldosterone system

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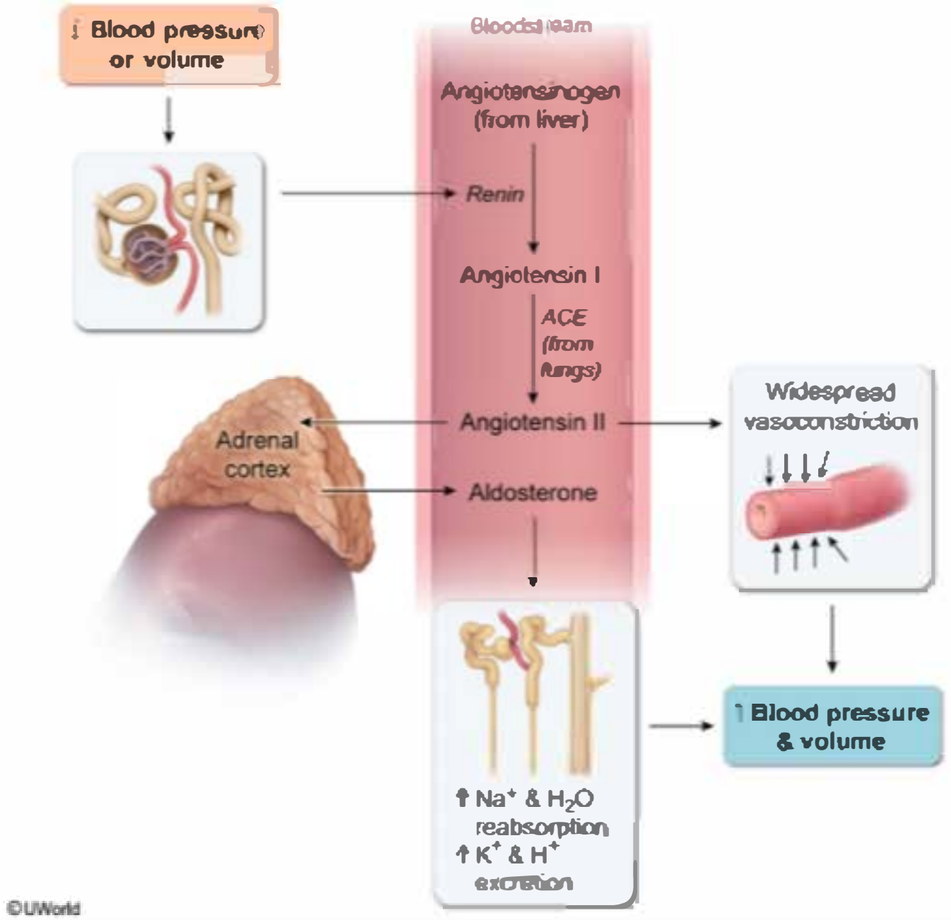
Educational object
Portal hypertension
activation of the ren

Exhibit Display

Renin-angiotensin system (RAS)



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A 3-month-old boy is evaluated for large-volume diarrhea and failure to thrive. The patient was born via spontaneous vaginal delivery at 38 weeks gestation. His mother reports that the pregnancy was uncomplicated and there is no family history of gastrointestinal disease. Physical examination shows mild abdominal distension, muscle wasting, and pitting edema. Histochemical evaluation reveals absence of enteropeptidase activity in the proximal intestinal villi. Activity of which of the following substances is most likely impaired in this patient?

A. Amylase
B. Bile salts
C. Lactase
D. Pepsin
E. Secretin
F. Trypsin

Submit

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- A. Amylase (2%)
- B. Bile salts (1%)
- C. Lactase (13%)
- D. Pepsin (10%)
- E. Secretin (3%)
- ✓ F. Trypsin (69%)

Omitted
Correct answer
F

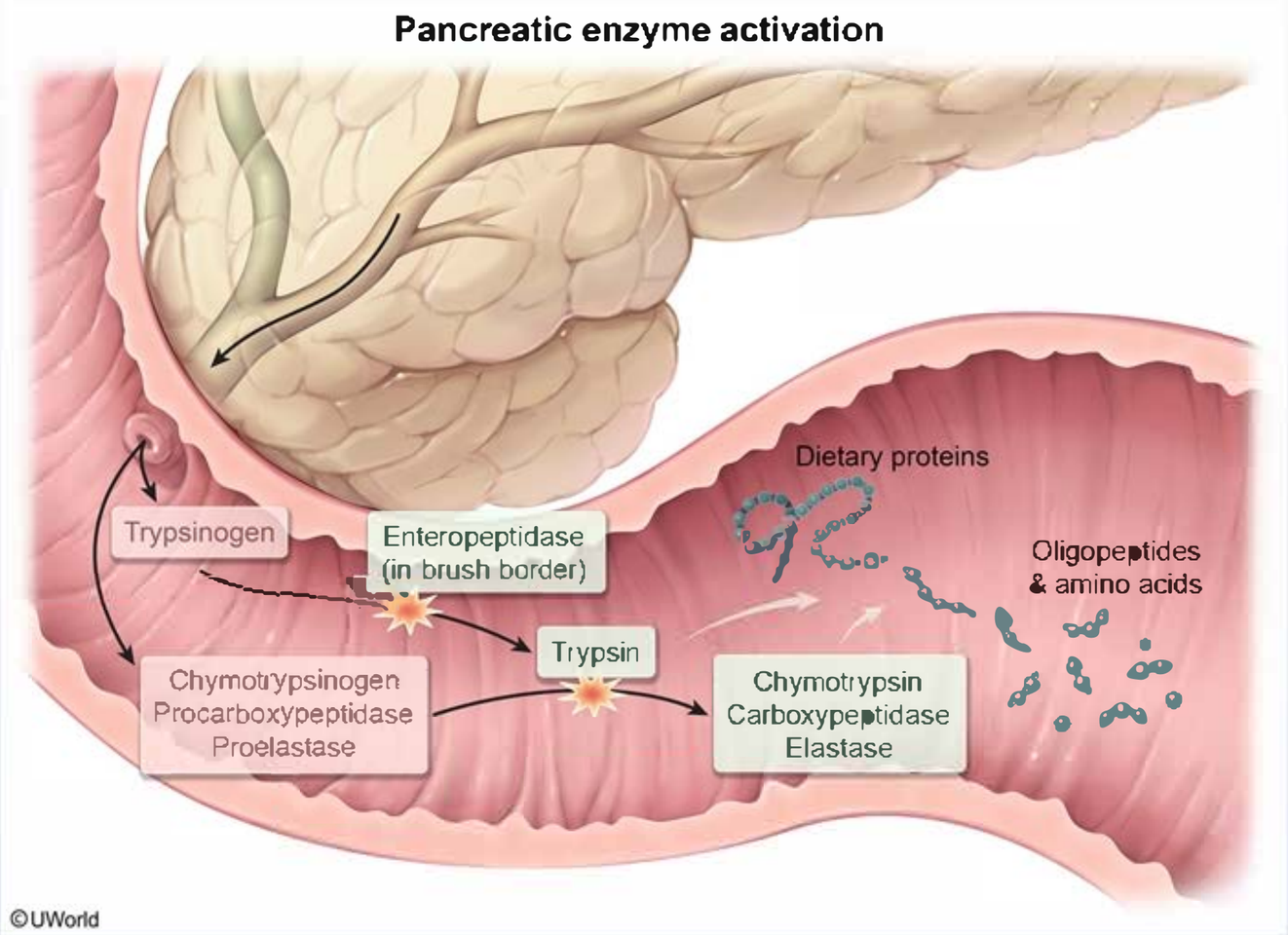
69%
Answered correctly

02 secs
Time Spent

04/20/2020
Last Updated

Explanation

Pancreatic enzyme activation



Although chemical digestion of proteins begins in the stomach, the majority of enzymatic breakdown and absorption occurs in the small intestine. Dietary proteins exist predominately as polypeptides; in order to be absorbed, they must be hydrolyzed to tripeptides, dipeptides, and amino acids. Hydrolysis begins in the stomach with the activation of pepsin by hydrochloric acid; it continues in the proximal small intestine, catalyzed by the pancreatic enzymes trypsin, chymotrypsin, carboxypeptidase, and elastase.

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Pancreatic enzymes, including trypsin, are secreted in their inactive forms. **Enteropeptidase**, a duodenal brush border enzyme, is responsible for **activation of trypsin** from its inactive precursor, trypsinogen. Once active, trypsin functions to cleave peptide bonds in dietary proteins and activate the other pancreatic enzymes. Enteropeptidase **deficiency** leads to both **protein and fat malabsorption** as trypsin is required to activate enzymes required for both lipid and protein digestion. The disease causes **diarrhea, failure to thrive, and edema** (due to hypoproteinemia).

(Choice A) Amylase is produced by the salivary glands and pancreas; it catalyzes the hydrolysis of complex carbohydrates to simple sugars.

(Choice B) Bile salts are formed through bile acid conjugation with amino acids glycine and taurine. This increases their solubility, allowing them to aid in lipid digestion and absorption through the formation of micelles.

(Choice C) Lactase is produced by the intestinal brush border and is responsible for the hydrolysis of lactose to glucose and galactose. Lactase non-persistence commonly develops during mid-childhood, resulting in lactose intolerance.

(Choice D) Chief cells of the stomach release the inactive form of pepsin, pepsinogen. Exposure to hydrochloric acid in the stomach activates pepsin, initiating protein digestion.

(Choice E) Secretin is a peptide hormone secreted by the S cells of the duodenum; it stimulates the secretion of bicarbonate from the pancreas and inhibits gastric acid secretion in the stomach. Neutralization of the acidic pH of food entering the duodenum from the stomach is necessary for the proper function of pancreatic enzymes.

Educational objective:
The duodenal brush border enzyme enteropeptidase activates trypsin from its inactive precursor, trypsinogen. Trypsin degrades complex polypeptides to dipeptides, tripeptides, and amino acids while activating other pancreatic enzymes. Enteropeptidase deficiency impairs both protein and fat absorption, leading to diarrhea, failure to thrive, and hypoproteinemia.

Pancreatic enzymes, including trypsin, are secreted in their inactive forms. **Enteropeptidase**, a duodenal brush border enzyme, is responsible for **activation of trypsin** from its inactive precursor, trypsinogen. Once active, trypsin functions to cleave peptide bonds in dietary proteins and activate the other pancreatic enzymes. Enteropeptidase **deficiency** leads to both **protein and fat malabsorption** as trypsin is required to activate enzymes required for both lipid and protein digestion. The disease causes **diarrhea, failure to thrive, and edema** (due to hypoproteinemia).

(Choice A) Amylase is produced by the salivary glands and pancreas; it catalyzes the hydrolysis of complex carbohydrates to simple sugars.

(Choice B) Bile salts are formed through bile acid conjugation with amino acids glycine and taurine. This increases their solubility, allowing them to aid in lipid digestion and absorption through the formation of micelles.

(Choice C) Lactase is produced by the intestinal brush border and is responsible for the hydrolysis of lactose to glucose and galactose. Lactase non-persistence commonly develops during mid-childhood, resulting in lactose intolerance.

(Choice D) Chief cells of the stomach release the inactive form of pepsin, pepsinogen. Exposure to hydrochloric acid in the stomach activates pepsin, initiating protein digestion.

(Choice E) Secretin is a peptide hormone secreted by the S cells of the duodenum; it stimulates the secretion of bicarbonate from the pancreas and inhibits gastric acid secretion in the stomach. Neutralization of the acidic pH of food entering the duodenum from the stomach is necessary for the proper function of pancreatic enzymes.

Educational objective:
The duodenal brush border enzyme enteropeptidase activates trypsin from its inactive precursor, trypsinogen. Trypsin degrades complex polypeptides to dipeptides, tripeptides, and amino acids while activating other pancreatic enzymes. Enteropeptidase deficiency impairs both protein and fat absorption, leading to diarrhea, failure to thrive, and hypoproteinemia.

Physiology	Gastrointestinal & Nutrition	Malabsorption
Subject	System	Topic

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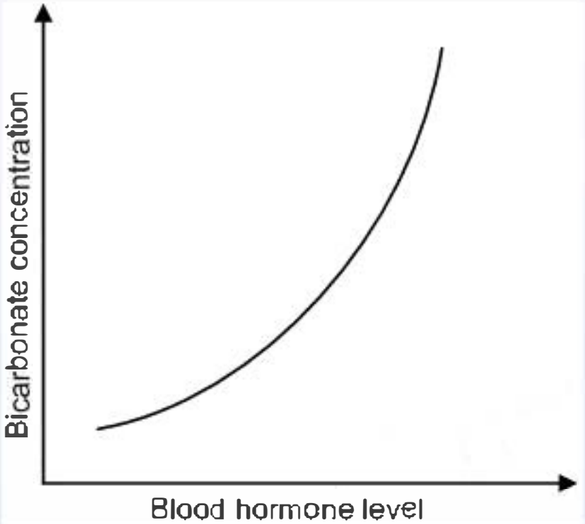
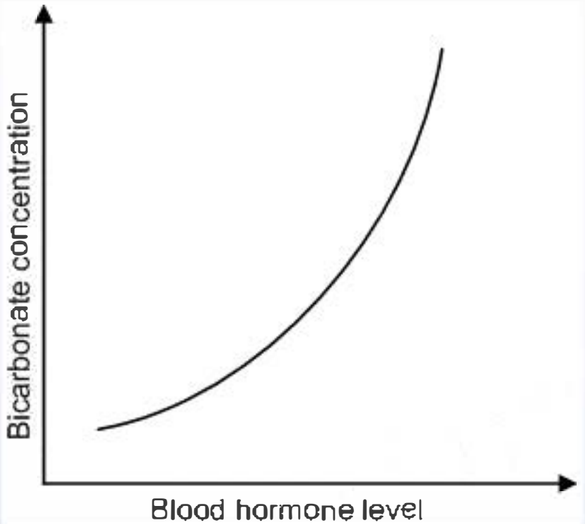
A 50-year-old man with a remote history of alcohol dependence is evaluated in the clinic for chronic diarrhea and weight loss. The patient has had bloating with daily loose stools for the past 2 years. His stool is described as greasy and malodorous. Pancreatic insufficiency is suspected despite normal abdominal imaging. The patient undergoes a test in which bicarbonate concentration is measured from duodenal aspirates as hormone A is infused intravenously. The data is plotted in the graph below.



Hormone A is most likely produced by which of the following cell types?

A. Duodenal S cells
B. Gastric G cells
C. Pancreatic alpha cells
D. Pancreatic beta cells
E. Parenchymal hepatocytes
F. Renal peritubular cells



Hormone A is most likely produced by which of the following cell types?

- ✓ A. Duodenal S cells (81%)
- B. Gastric G cells (9%)
- C. Pancreatic alpha cells (5%)
- D. Pancreatic beta cells (1%)
- E. Parenchymal hepatocytes (0%)
- F. Renal peritubular cells (0%)

Omitted
Correct answer

81%
Answered correctly

05 secs
Time Spent

06/26/2020
Last Updated

Important gastrointestinal hormones

Hormone	Actions	Secretion site
Gastrin	• ↑ Gastric H^+ secretion	G cells (gastric antrum, duodenum)
Somatostatin	• ↓ Secretion of most GI hormones	D cells (pancreatic islets, gut mucosa)
Cholecystokinin	• ↑ Pancreatic enzyme & HCO_3^- secretion	I cells (small intestine)
Secretin	• ↑ Pancreatic HCO_3^- secretion • ↓ Gastric H^+ secretion	S cells (small intestine)
GIP	• ↑ Insulin release • ↓ Gastric H^+ secretion	K cells (small intestine)
Motilin	• ↑ GI motility	M cells (small intestine)

GI = gastrointestinal; GIP = gastric inhibitory peptide; H^+ = hydrochloric acid; HCO_3^- = bicarbonate.

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Secretin is a hormone produced by **intestinal S cells** that **increases bicarbonate secretion** from the pancreas. Secretin stimulates the **exocrine pancreatic ductal cells** to release a high volume of **bicarbonate-rich and chloride-poor fluid** into the small bowel. The release of gastric acid into the duodenum is the most potent stimulus for secretin release, which begins as the duodenal pH drops below 5 and rises

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(Choice B) Gastrin is the major hormone secreted by G cells of the gastric antrum. Gastrin increases parietal cell acid production and therefore increases the amount of acid delivered from the stomach to the duodenum.

(Choices C and D) The endocrine pancreas is composed of glucagon-producing alpha cells, insulin-producing beta cells, and somatostatin-producing D cells. Insulin and glucagon help regulate blood glucose levels and metabolic homeostasis. Somatostatin inhibits the secretion of insulin, glucagon, gastrin, and most other gastrointestinal hormones.

(Choice E) The liver parenchyma is not a significant source of circulating hormones that affect pancreatic exocrine function.

(Choice F) Renal peritubular cells are responsible for producing erythropoietin, a hormone that stimulates red blood cell production in the bone marrow.

Educational objective:
Secretin is produced by S cells in the duodenal mucosa in response to stimulation by intraluminal acidity. Secretin stimulates the release of bicarbonate-rich secretions from the exocrine pancreas, which is the major source of acid-neutralizing bicarbonate entering the duodenum.

References

- Secretin, 100 years later.
- Neurohormonal control of exocrine pancreas.

Physiology | Gastrointestinal & Nutrition | Gastrointestinal hormones

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Physiology	Gastrointestinal & Nutrition	Gastrointestinal hormones
Subject	System	Topic

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A 42-year-old man comes to the office due to leg swelling. He has also had yellowing of the eyes and progressive abdominal distension for the past 3 weeks, as well as an associated 7-kg (15.4-lb) weight gain. The patient drinks at least 8 beers a day and in the past has been admitted to the hospital for intoxication and seizures. He denies smoking or intravenous drug use. Vital signs are within normal limits. Scleral icterus, spider angiomas, and palmar erythema are present. There is no jugular venous distension, and cardiac auscultation reveals a normal rate and rhythm. Breath sounds are normal. The abdomen is diffusely enlarged, and a fluid wave is easily elicited. Pitting edema is present in the bilateral lower extremities. Which of the following pathophysiologic changes is most likely present in this patient?

	Plasma oncotic pressure	Splanchnic vascular resistance	Effective arterial blood volume	Capillary permeability
A.	↓	↓	↓	Normal
B.	Normal	↑	↓	Normal
C.	↓	Normal	↑	↑
D.	Normal	Normal	Normal	↑
E.	↑	↓	Normal	Normal

Submit

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B.	Normal	↑	↓	Normal
C.	↓	Normal	↑	↑
D.	Normal	Normal	Normal	↑
E.	↑	↓	Normal	Normal

Submit

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	Plasma oncotic pressure	Splanchnic vascular resistance	Effective arterial blood volume	Capillary permeability
✓ A. (63%)	↓	↓	↓	Normal
B. (6%)	Normal	↑	↓	Normal
C. (24%)	↓	Normal	↑	↑
D. (1%)	Normal	Normal	Normal	↑
E. (3%)	↑	↓	Normal	Normal

Omitted
Correct answer
A

63%
Answered correctly

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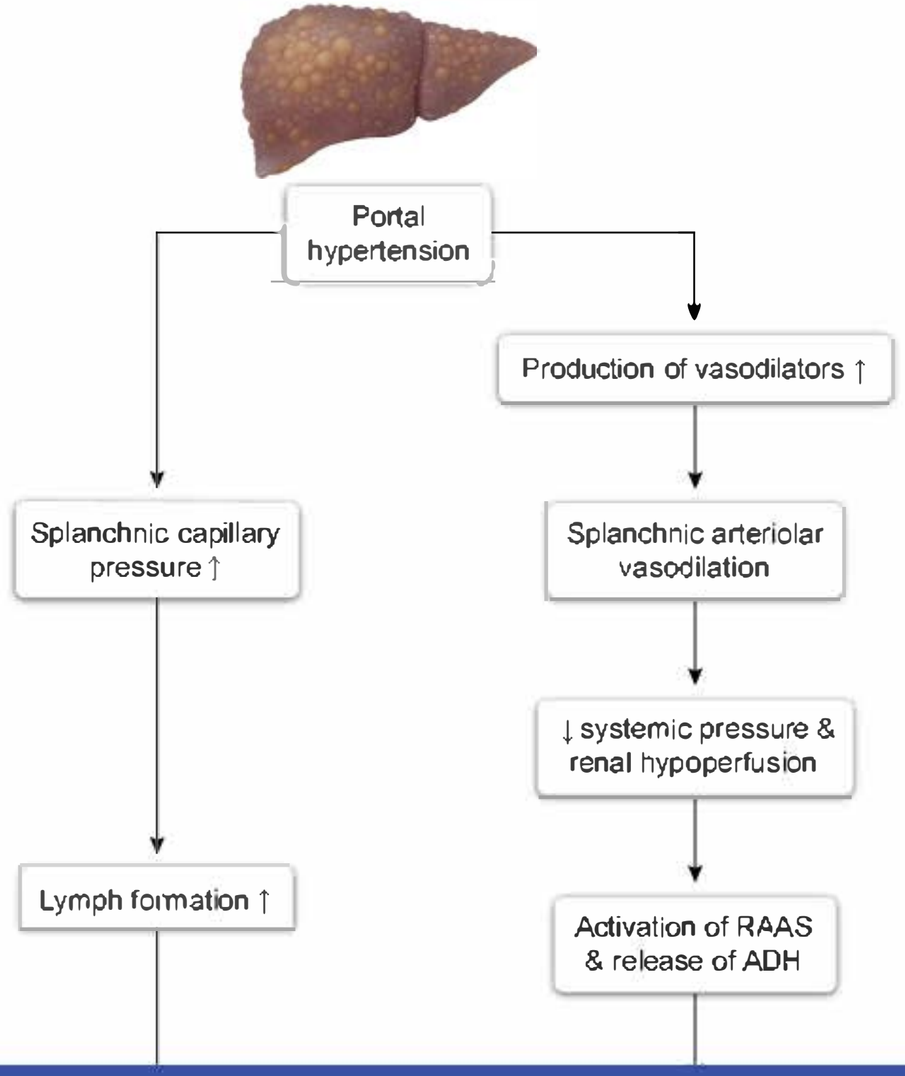
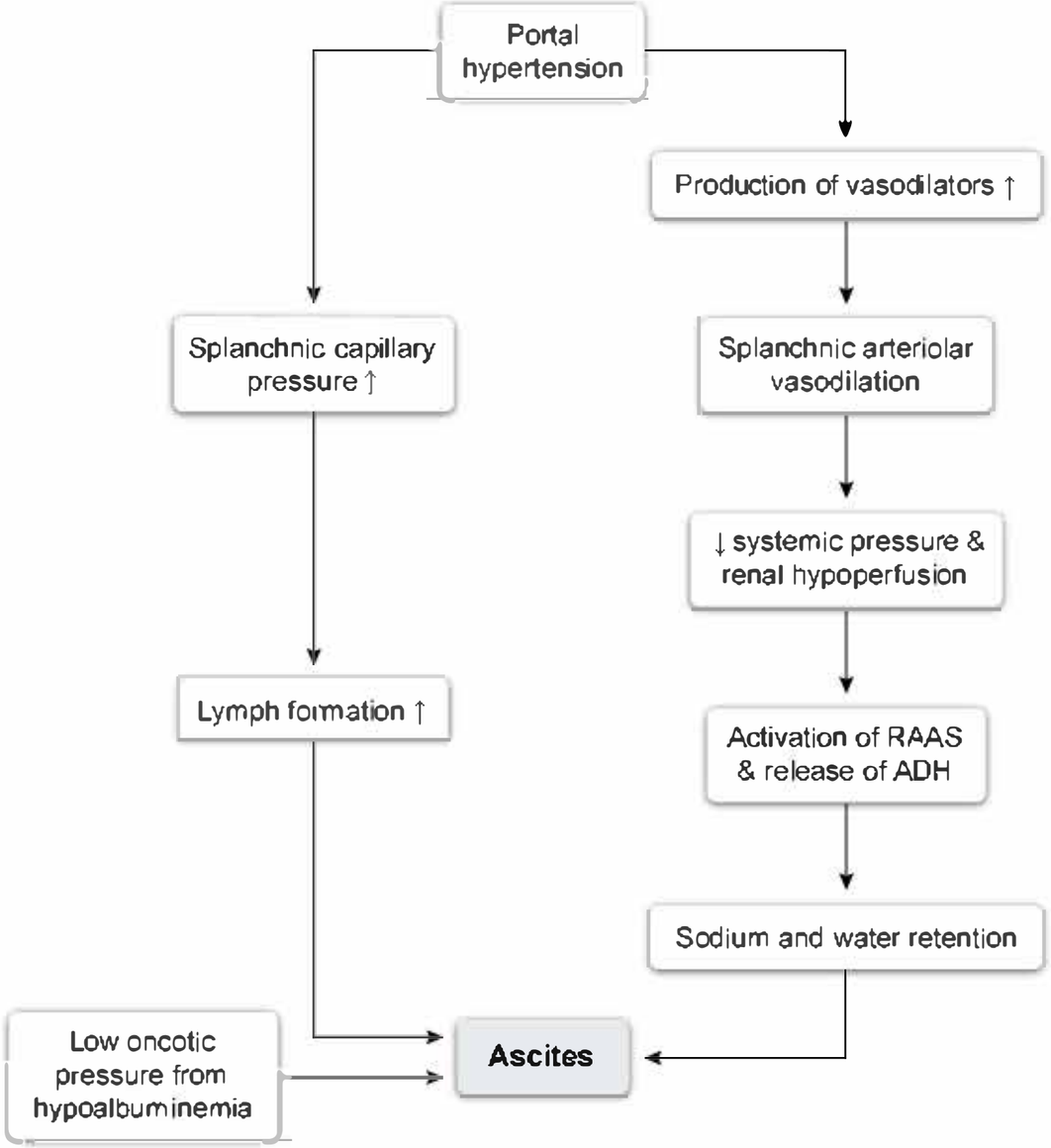
Time Spent

03/26/2020
Last Updated

Pathogenesis of ascites in cirrhosis




Portal hypertension
Production of vasodilators ↑
Splanchnic capillary pressure ↑
Splanchnic arteriolar vasodilation
↓ systemic pressure & renal hypoperfusion
Lymph formation ↑
Activation of RAAS & release of ADH
Sodium and water retention
Low oncotic pressure from hypoalbuminemia
Ascites

This patient has **ascites**, characterized by the accumulation of fluid within the peritoneal cavity. Ascites may occur in a variety of diseases (eg, peritoneal metastasis, congestive heart failure); however, in this patient with a history of heavy alcohol use and stigmata of chronic liver disease (eg, jaundice, spider angiomas, palmar erythema), it likely occurred from **cirrhosis**.

Cirrhosis is characterized by progressive liver fibrosis, which results in the formation of a high-resistance system (ie, **portal hypertension**). This results in the following alterations:

1. Nitric oxide is released, possibly due to stimulation by bacterial products (eg, endotoxin), which can more easily translocate from the gastrointestinal tract due to reduced host defenses (eg, impaired reticuloendothelial function) and portosystemic shunt formation (eg, decreased toxin clearance).
2. Splanchnic vasodilation occurs as a result, which **decreases the splanchnic vascular resistance** and **lowers the effective arterial blood volume** (EABV) due to blood pooling in the splanchnic vascular bed.
3. Low perfusion pressure results in activation of the renin-angiotensin-aldosterone system along with increased secretion of antidiuretic hormone, leading to **retention of sodium and water**.

Additionally, cirrhosis results in impaired synthetic function, leading to hypoalbuminemia and **low plasma oncotic pressure**, which reduces fluid resorption from the interstitium. Paired with portal hypertension (ie, increased hydrostatic pressure), these changes result in a vicious cycle of hypervolemia and third spacing, promoting the formation of ascites and peripheral edema. The capillaries remain physiologically normal, so **permeability is unchanged**.

(Choice B) Congestive heart failure causes low EABV (due to low cardiac output) with a consequent increase in sympathetic tone that results in increased splanchnic vascular resistance. Oncotic pressure and capillary permeability are unaffected.

(Choice C) Nephrotic syndrome causes edema due to increased permeability of the glomerular capillary wall, leading to loss of albumin, which decreases capillary oncotic pressure. Most patients also have a defect in sodium excretion from the kidneys, resulting in sodium retention and increased EABV. Splanchnic vascular resistance is unaffected.

(Choice D) Localized allergic reactions (eg, insect sting) can lead to increased capillary permeability, leading to dermal edema. Oncotic pressures, splanchnic vascular resistance, and EABV are unaffected.

(Choice E) Increased capillary oncotic pressure tends to inhibit fluid accumulation in the interstitial spaces, thereby decreasing peripheral edema

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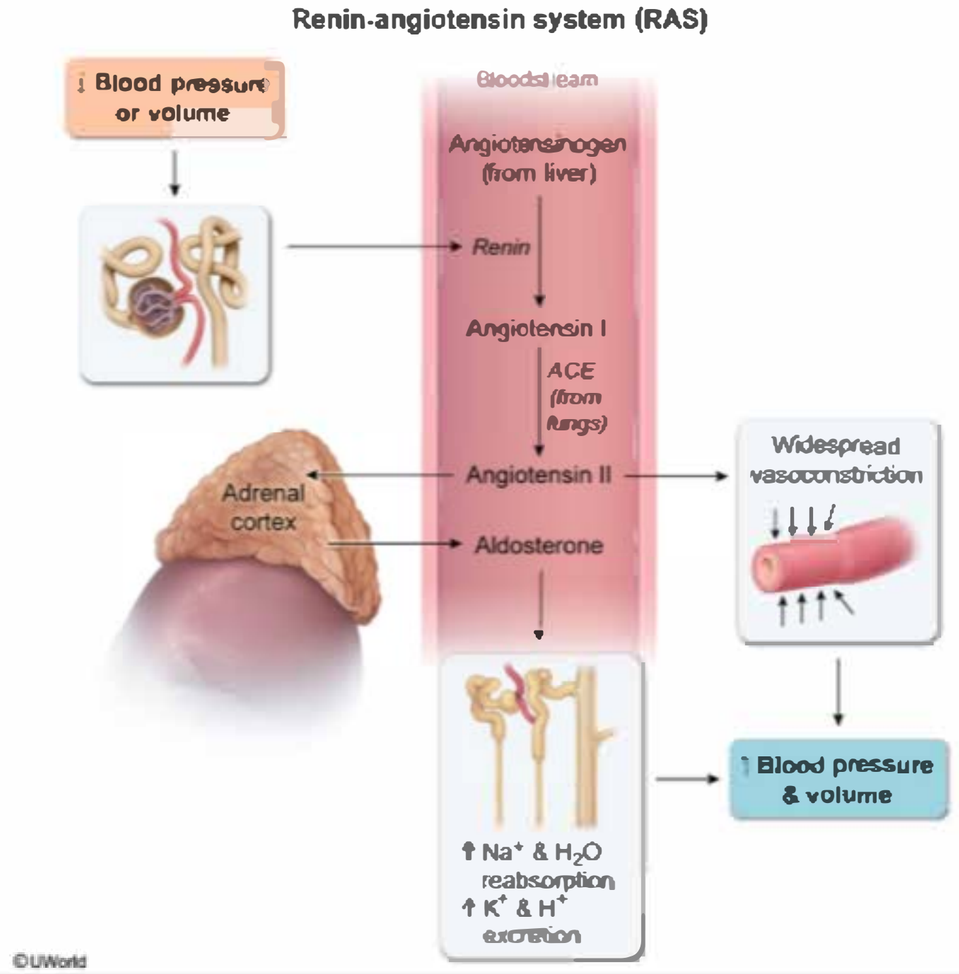
Exhibit Display



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(Choice E) Increased capillary oncotic pressure tends to inhibit fluid accumulation in the interstitial spaces, thereby decreasing peripheral edema and ascites.

Educational objective:

Ascites in cirrhosis develops from hemodynamic changes related to portal hypertension. Splanchnic vasodilation decreases the splanchnic vascular resistance and lowers the effective arterial blood volume, which causes activation of the renin-angiotensin-aldosterone system and promotes sodium and water retention. Additionally, low oncotic pressure (ie, hypoalbuminemia due to impaired hepatic function) reduces fluid resorption from the interstitium.

References

- Fluid retention in cirrhosis: pathophysiology and management.

Physiology	Gastrointestinal & Nutrition	Ascites
Subject	System	Topic

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Experimental gastric function monitoring in healthy subjects before, during and after a meal shows an initial rise, peak, and then decline in gastric acid production. Which of the following helps most to down-regulate gastric secretion after a meal?

A. Basal secretion

B. Cephalic phase

C. Gastrin release

D. Intestinal influences

E. Receptive relaxation

F. Postprandial alkaline tide

Submit

Experimental gastric function monitoring in healthy subjects before, during and after a meal shows an initial rise, peak, and then decline in gastric acid production. Which of the following helps most to down-regulate gastric secretion after a meal?

- A. Basal secretion (3%)
- B. Cephalic phase (4%)
- C. Gastrin release (10%)
- ✓ D. Intestinal influences (60%)
- E. Receptive relaxation (5%)
- F. Postprandial alkaline tide (15%)

Omitted
Correct answer
D

60%
Answered correctly

02 secs
Time Spent

04/22/2020
Last Updated

Explanation

Classically, the stimulation of acid secretion within the stomach is separated into three phases: cephalic, gastric, and intestinal. The cephalic phase is mediated primarily by cholinergic and vagal mechanisms, and is triggered by the thought, sight, smell, and taste of food. The gastric phase is mediated by the presence of gastrin (which stimulates histamine secretion and therefore, indirectly, acid secretion), and is triggered by the chemical stimulus of food and distension of the stomach. The intestinal phase is initiated when protein-containing food enters the duodenum, but this phase plays only a minor role in stimulating gastric acid secretion.

In fact, intestinal influences are effective in down-regulating gastric acid secretion after a meal. The ileum and colon release peptide YY, which

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In fact, intestinal influences are effective in down-regulating gastric acid secretion after a meal. The ileum and colon release peptide YY, which binds to receptors on the endocrine, histamine-containing cells described as enterochromaffin-like (ECLs). Such binding counteracts the cephalic and gastric phases of acid secretion by inhibiting gastrin-stimulated histamine release from ECLs. Other factors that inhibit acid secretion include somatostatin and prostaglandins.

(Choice A) Basal gastric secretion has been defined as the gastric juices secreted in the absence of intentional or avoidable stimuli (essentially, the "baseline" gastric acid secretion). Basal gastric secretion is not an important factor in the down-regulation of postprandial gastric secretion.

(Choices B and C) The cephalic and gastric phases both stimulate gastric acid secretion, not down-regulate it.

(Choice E) Receptive relaxation is a reflex that allows the gastric fundus to dilate in anticipation of food passing through the pharynx and esophagus. It is not an important factor in the down-regulation of postprandial gastric secretion.

(Choice F) Postprandial alkaline tide is defined as an increase in plasma HCO_3^- and decrease in plasma Cl^- secondary to the surge of acid within the gastric lumen. It is not an important factor in the down-regulation of postprandial gastric secretion.

Educational Objective:
The cephalic and gastric phases stimulate gastric acid secretion, while intestinal influences tend to reduce gastric acid secretion.

Physiology	Gastrointestinal & Nutrition	Gastric physiology
Subject	System	Topic

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A 1-monthold, full-term African-American boy is brought to the office for a routine checkup. He is exclusively breastfed. The boy does not have any medical problems, and his hospital stay after birth was unremarkable. His mother consumes a well-balanced diet, takes no medications, and does not drink alcohol. The boy is at the 50th percentile for weight and the 60th percentile for height. His physical examination is normal. At this time, supplementation with which of the following should be recommended for this infant?

A. Folic acid

B. Iron

C. Riboflavin

D. Thiamine

E. Vitamin A

F. Vitamin D

Submit

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- A. Folic acid (4%)
- B. Iron (15%)
- C. Riboflavin (2%)
- D. Thiamine (3%)
- E. Vitamin A (5%)
- ✓ F. Vitamin D (68%)

Omitted
Correct answer
F

68%
Answered correctly

01 sec
Time Spent

01/28/2020
Last Updated

Explanation

Supplementation for breastfed infants	
Supplement	Risk factors

Supplementation for breastfed infants	
Supplement	**Risk factors**
Vitamin D	• Exclusive breastfeeding • Lack of sunlight exposure • Dark skin pigmentation
Iron	• Preterm/lowbirthweight

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Breast milk is the gold standard of infant nutrition; it contains proteins, carbohydrates, fats, vitamins, trace minerals, immunoglobulins, amino acids, and enzymes. All vitamins and trace minerals are present in adequate amounts **except vitamins D and K**. Vitamin K is supplemented by an intramuscular injection at delivery to prevent hemorrhagic disease in the newborn.

Regular sunlight exposure typically provides sufficient vitamin D. However, infants are generally shielded from direct sunlight due to sunburn risk. In addition, infants with dark skin pigmentation require more sunlight exposure to produce adequate vitamin D. Melanin is a natural sunblock and prevents ultraviolet rays from reaching the skin for vitamin D synthesis. Prolonged vitamin D insufficiency can lead to rickets (inadequate bone and cartilage mineralization). Therefore, **all exclusively breastfed** infants should receive **vitamin D supplementation** to **prevent rickets**. Formula-fed infants do not require supplementation as formula is fortified with adequate amounts of vitamin D.

(Choices A, C, and D) Human breast milk normally provides adequate amounts of folic acid, riboflavin, and thiamine for full-term infants.

(Choice B) Although breast milk has low iron content, the bioavailability of iron in breastmilk is considerably higher than supplemental iron and is sufficient for infants until age 4 months. Due to decreasing iron concentration in breast milk over time, supplementation is recommended for breastfed infants at age >4 months until solid food intake provides an adequate amount.

(Choice E) Vitamin A stores in the liver are low at birth but rapidly increase due to the large amount in colostrum and breast milk.

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Educational objective:
The breast milk content of vitamins D and K is typically insufficient for the nutritional needs of the newborn. Vitamin K is given parenterally to all newborns at birth to prevent hemorrhagic disease of the newborn. Exclusively breastfed infants require vitamin D supplementation due to lack of sunlight exposure and to prevent rickets.

References

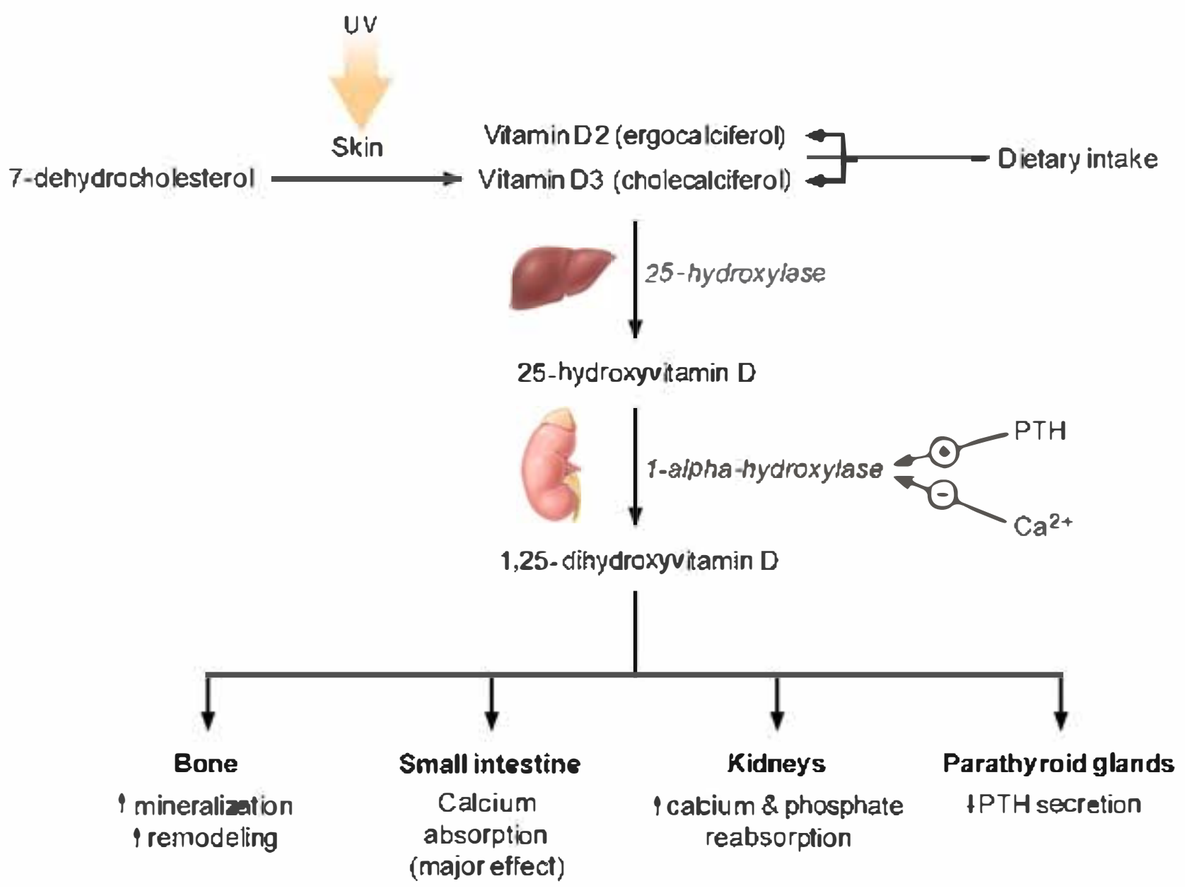
- Is vitamin D supplementation necessary in healthy full-term breastfed infants? A follow-up study of bone mineralization in healthy full-term infants with and without supplemental vitamin D.
- Adherence to vitamin D recommendations among US infants.
- Prevalence and risk factors for vitamin D deficiency among healthy infants and young children in Sacramento, California.

Physiology Gastrointestinal & Nutrition Breastfeeding

Breast milk is the gold standard of infant nutrition; it contains proteins, carbohydrates, fats, vitamins, trace minerals, immunoglobulins, amino

Exhibit Display

Normal vitamin D metabolism



PTH = parathyroid hormone.

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Physiology | Gastrointestinal & Nutrition | Breastfeeding

A 27-year-old woman in her 2nd trimester of pregnancy comes to the office due to constipation. Her stools have become increasingly hard and pellet-like over the past few weeks; bowel movements before then were always regular. She also has mild crampy abdominal pain and bloating, both of which improve after defecation. Medical history and family history are both unremarkable. This is the patient's first pregnancy and has so far been uncomplicated. On physical examination, the abdomen appears gravid but nontender. Which of the following best explains this patient's symptoms?

A. Decreased colonic smooth muscle activity

B. Decreased parasympathetic input to the colon

C. Impaired relaxation of pelvic floor muscles

D. Thickening of intestinal secretions

E. Thinning of the rectovaginal septum

Submit

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- A. Decreased colonic smooth muscle activity (36%)
- B. Decreased parasympathetic input to the colon (16%)
- C. Impaired relaxation of pelvic floor muscles (33%)
- D. Thickening of intestinal secretions (8%)
- E. Thinning of the rectovaginal septum (5%)

Omitted
Correct answer
A

36%
Answered correctly

02 secs
Time Spent

05/12/2020
Last Updated

Explanation

Common gastrointestinal adaptations in pregnancy		
	Adaptation	Complication
Esophagus	• Progesterone decreases LES tone	• GERD

Common gastrointestinal adaptations in pregnancy		
	Adaptation	Complication
Esophagus	• Progesterone decreases LES tone	• GERD
Gallbladder	• Estrogen increases cholesterol secretion into bile • Progesterone reduces gallbladder motility	• Cholelithiasis • Cholecystitis
Intestines	• Progesterone decreases colonic smooth muscle activity and decreases fasting MMCs in the small intestine • Gravid uterus mechanically impedes small intestine transit	• Constipation • Bloating • Flatulence
Rectum	• Gravid uterus increases venous pressure	• Hemorrhoids

LES = lower esophageal sphincter; GERD = gastroesophageal reflux; MMC = migrating myoelectric complex.

Pregnancy has multiple effects in the gastrointestinal tract due to increasing levels of progesterone and estrogen as well as the effects of a gravid uterus on surrounding structures. **Constipation** is particularly common in pregnant patients and is largely due to the effects of **progesterone**, which has been shown to **reduce colonic smooth muscle activity**. Progesterone also prevents the release of motilin, a hormone responsible for stimulating the fasting migrating myoelectric complex, which normally helps propel undigested food from the small intestines into the colon. Constipation may be exacerbated in later stages of pregnancy as the gravid uterus increases pressure on the small intestine, slowing intestinal transit.

Other common gastrointestinal alterations in pregnancy include reduced lower esophageal sphincter pressure (promoting gastroesophageal reflux) and gallbladder stasis (increasing the likelihood of developing gallstones). Hemorrhoids are also common and result from a rise in pressure within the rectal venous system due to the gravid uterus.

(Choice B) The parasympathetic nervous system stimulates peristalsis and gastrointestinal motility. Reduced parasympathetic input to the colon can lead to constipation; however, this typically occurs with neurologic disorders (eg, spinal cord injury, multiple sclerosis).

(Choice C) Defecatory dysfunction results from an inability to relax pelvic floor muscles, which prevents proper evacuation of stool from the rectal vault. [illegible]

Common gastrointestinal adaptations in pregnancy		
	Adaptation	Complication
Esophagus	• Progesterone decreases LES tone	• GERD
Gallbladder	• Estrogen increases cholesterol secretion into bile • Progesterone reduces gallbladder motility	• Cholelithiasis • Cholecystitis
Intestines	• Progesterone decreases colonic smooth muscle activity and decreases fasting MMCs in the small intestine • Gravid uterus mechanically impedes small intestine transit	• Constipation • Bloating • Flatulence
Rectum	• Gravid uterus increases venous pressure	• Hemorrhoids

LES = lower esophageal sphincter; GERD = gastroesophageal reflux; MMC = migrating myoelectric complex.

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(Choice B) The parasympathetic nervous system stimulates peristalsis and gastrointestinal motility. Reduced parasympathetic input to the colon can lead to constipation; however, this typically occurs with neurologic disorders (eg, spinal cord injury, multiple sclerosis).

(Choice C) Defecatory dysfunction results from an inability to relax pelvic floor muscles, which prevents proper evacuation of stool from the rectal vault. Although this can occur after trauma (eg, traumatic vaginal delivery), it is not a physiologic change that occurs during pregnancy.

(Choice D) Thickened intestinal secretions can cause constipation; however, this is typically seen in patients with cystic fibrosis, which affects water and electrolyte transportation. Patients with cystic fibrosis are typically diagnosed in infancy and have recurrent respiratory infections and failure to thrive.

(Choice E) Rectoceles occur when the rectum herniates into the vaginal canal through a thinned rectovaginal septum. Although they also cause constipation, rectoceles typically occur in elderly individuals with a history of multiple vaginal deliveries, not during pregnancy. Patients often report needing to manually compress the vagina to induce bowel movements.

Educational objective:
Constipation is common during pregnancy and results from the inhibitory effect of progesterone on colonic smooth muscle contractions and the fasting migrating myoelectric complex. Other common pregnancy-related changes of the gastrointestinal system include decreased lower esophageal sphincter pressure (leading to gastroesophageal reflux) and gallbladder stasis (leading to gallstone formation).

Physiology	Gastrointestinal & Nutrition	Constipation
Subject	System	Topic

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Many drugs and toxins act by inhibiting substance flow across cell membranes. Which of the following inhibits primary active transport?

A. Tetrodotoxin

B. Verapamil

C. Omeprazole

D. Lidocaine

E. Dofetilide

Submit

Many drugs and toxins act by inhibiting substance flow across cell membranes. Which of the following inhibits primary active transport?

A. Tetrodotoxin (12%)

B. Verapamil (5%)

✓ C. Omeprazole (70%)

D. Lidocaine (7%)

E. Dofetilide (3%)

Omitted
Correct answer
C

70%
Answered correctly

02 secs
Time Spent

02/21/2020
Last Updated

Explanation

Transport of ions or molecules across cell membranes can be either passive (along the concentration gradient) or active (against the concentration gradient). The former does not require energy, while the latter utilizes energy from ATP. In primary active transport, the carrier molecules are enzymes that hydrolyze ATP (ATPases) and use the energy released to transport ions and molecules against their concentration gradients. Examples of such carriers are the Na/K ATPase, the H/K ATPase, and the Ca ATPase.

Synthesis of HCl by parietal cells of the gastric mucosa is dependent on the H/K ATPase, which is known as a proton pump. This carrier transports hydrogen ions into the gastric lumen in exchange for potassium ions.

Omeprazole and other proton pump inhibitors inhibit the H/K ATPase, thus decreasing the concentration of HCl in the gastric lumen. These medications are used for treatment of peptic ulcer disease, gastroesophageal reflux disease (GERD), and diseases associated with increased

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(Choice A) Tetrodotoxin, a potent neurotoxin found in pufferfish, functions by blocking the voltage-gated sodium channels in nerve cell membranes. It inhibits passive transport of sodium.

(Choice B) Verapamil blocks L-type calcium channels, thus inhibiting passive transport of calcium ions in cardiomyocytes.

(Choice D) Lidocaine is a local anesthetic and a class IB antiarrhythmic agent that functions by blocking voltage-gated sodium channels in sensory neurons, Purkinje fibers and ventricular cells.

(Choice E) Dofetilide is a class III antiarrhythmic drug that blocks passive transport of potassium, specifically the delayed outward rectifier potassium current, in cardiomyocytes leading to a prolongation of the refractory period and of the QT interval.

Educational Objective:
Parietal cells release hydrogen ions into the gastric lumen by means of the H/K ATPase, which requires hydrolysis of ATP and is therefore an active transport mechanism. Omeprazole and other proton pump inhibitors suppress the activity of the gastric parietal cell H/K ATPase leading to an increase in the pH of the gastric lumen.

Physiology	Gastrointestinal & Nutrition	Proton pump inhibitors
Subject	System	Topic

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